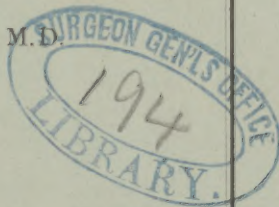


RANNEY (A.L.)

THE
CORPORA QUADRIGEMINA.

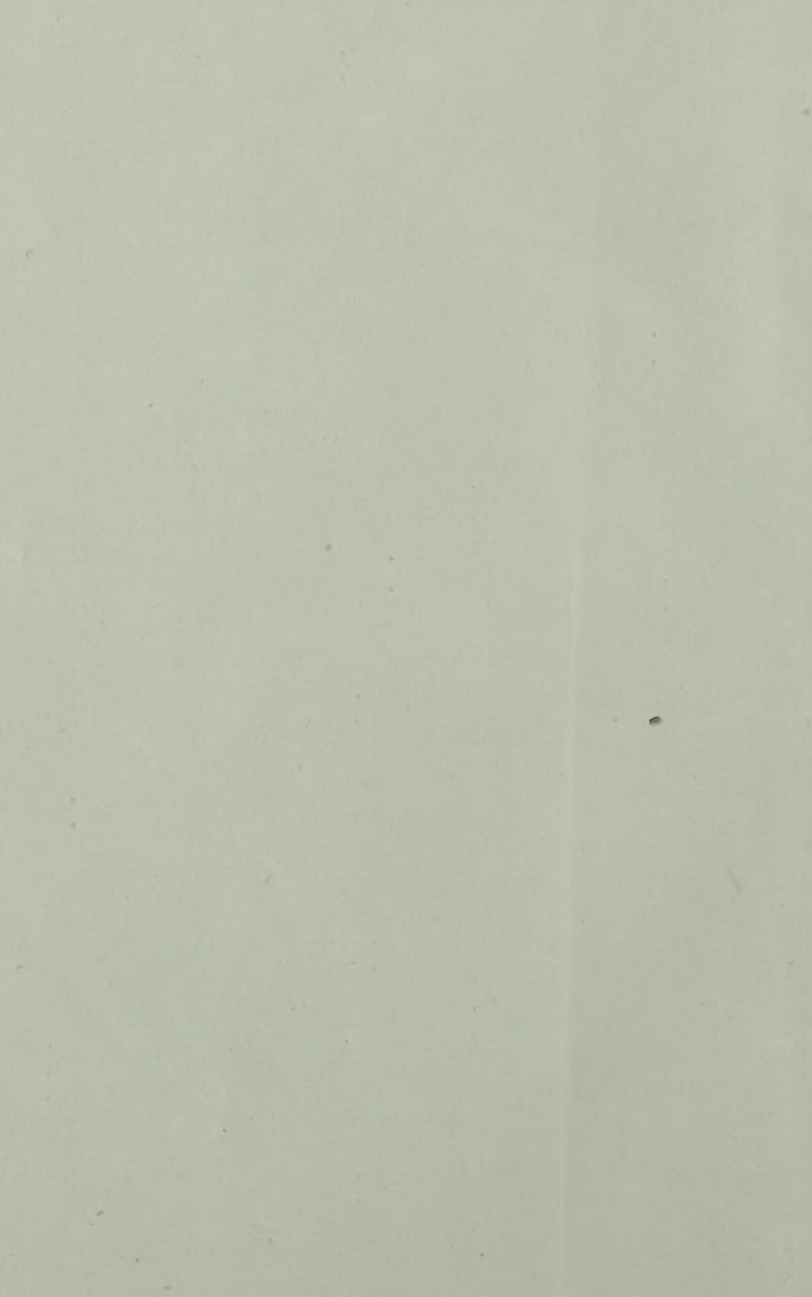
WITH
REMARKS CONCERNING THE DIAGNOSIS
AND LOCALIZATION OF LESIONS
AFFECTING SIGHT.

BY
AMBROSE L. RANNEY, M.D.



Reprinted from THE MEDICAL RECORD, August 18, 1883.

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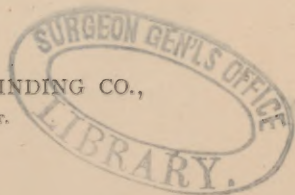
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WITH REMARKS CONCERNING THE DIAGNOSIS AND LOCALIZATION OF LESIONS AFFECTING SIGHT.¹

AMONG the investigators who have devoted special attention to these bodies may be prominently mentioned Adamuck, Knoll, Budge, Hensen, Voelkers, Flourens, Schiff, Ferrier, McKendrick, Gudden, and many others of note. Some have observed the effects of their removal in animals; others have studied the results of stimulation of their superficial and deep parts; while a few have recorded the results of destruction of the optic apparatus and certain convolutions of the cerebral hemispheres, as possessing a peculiar bearing upon points in dispute regarding these bodies. From these different sources a mass of evidence has been accumulated which appears in some instances to lead to contradictory conclusions. It is only by comparing the views of the investigators mentioned, and bringing to bear upon the subject what is also taught us by anatomical research, that the web may be partially disentangled.

The connection of the anterior quadrigeminal bodies, or the *nates cerebri*, with the optic tract and the sense of sight appears to be far more intimate than that of the posterior lobules, or *testes cerebri*, as was first pointed

¹ This article formed a part of my course of lectures during the winter of 1882-83 before the students of the Medical Department of the University of the City of New York.

out clearly by Gudden. This observer found that the extirpation of the eye on one side of a young animal was followed by a degeneration and atrophy of the nates cerebri and its brachium; the testis and its brachium remaining unaltered. This view is apparently sustained also by the fact that the mole has the testes cerebri largely developed, whereas the nates cerebri are markedly atrophied. Adamuck believed that he had clearly demonstrated the existence of a centre within the nates which presided over those movements of the eye and pupil which are essential to the *accommodation of vision for near objects*, as well as the *co-ordination of all ocular movements*. Knoll found, however, that reflex contractions of the pupil remained after removal of the corpora quadrigemina; and Hensen and Voelkers have been apparently successful in mapping out the topography of the centres which preside over ocular and pupillary movements with greater accuracy than their predecessors. They were able to produce at will, by carefully applied electric stimulation in the region of the floor of the aqueduct of Sylvius, independent movements of the eye and pupil. In the dog, upon which animal these experiments were made, a centre which governed the *accommodation of vision*¹ was found to be situated in the posterior part of the third ventricle near to the aqueduct, while a centre for pupillary contraction and one also for its dilatation were found in the front part of the floor of the aqueduct of Sylvius, the former lying in the median plane and the latter more to the sides. The same observers state that a centre, which governs those muscles of the eyeball which are supplied by the third cranial nerve, can be found in the floor of the aqueduct, immediately behind that which presides over pupillary contraction. Whether we accept these statements as dem-

¹ This centre manifested an apparent control over the *ciliary muscle* only, and created alterations in the antero-posterior measurement of the crystalline lens of the eye.

onstrated or not, we know positively that such centres exist somewhere, and are so associated in their action that, when the eyeballs are directed inward and downward, as for near vision, the pupils are at the same time contracted; and when the eyeballs are directed upward and returned to a state of parallelism, the pupils are dilated to a corresponding extent. On the contrary, when the eyeballs are moved sideways in unison, the pupils remain unchanged. A most positive proof that the pupillary movements are not of a psychical nature is afforded by the experiments of Adamuck, who produced movements of *both eyes* by stimulation of the corpora quadrigemina of either side, and who also observed that the pupils were at the same time made to perform their proper movements. When, however, the corpora quadrigemina of the two sides were separated by a median incision, stimulation of the centres of either side caused movements of the corresponding eyeball only. In both experiments, changing the seat of stimulation caused modifications of ocular movements.

It was only after Knoll had shown that the reflex movements of the pupils remained after complete excision of the corpora quadrigemina, and the discovery of Hensen and Voelkers that the effects of stimulation of these bodies, as first practised by Adamuck, were not uniform until the *underlying parts* were directly reached, that discrepancies between these observers were explained.

To determine the true relations which these bodies bear to the special sense of sight is perhaps one of the most difficult problems in physiology.

Flourens and many subsequent observers have shown us that unilateral extirpation of the corpora quadrigemina in mammals and birds leads to a blindness of the opposite eye; and even when the cerebral hemispheres are removed without disturbing these bodies, that an apparently crude vision still remains. We have many experiments, however, to show that destruction of certain convolutions

of the cerebrum also produced the most profound effects upon vision in spite of the undisturbed action of the quadrigeminal bodies. When we discussed the optic thalamus,¹ it was also stated that many clinical observations pointed toward the existence of a centre within that body which in some way modified or presided over visual impressions. We know also that lesions within the so-called "internal capsule" of the cerebrum frequently produce most serious impairment of vision, and conjugate deviation of the eyes.

Now, how are we to explain, theoretically, such contradictory phenomena? What views are we apparently justified in holding (from the standpoint of our present knowledge upon the subject) regarding the relations of the cerebral cortex, corpora quadrigemina, corpora geniculata, optic thalami, and internal capsule of the cerebrum, to the fibres of the optic tracts and the external organs of sight?

I think we are justified in attributing to the cells of the *cerebral cortex* or the external gray matter of the hemispheres alone our conceptions of the external world, as portrayed to us by means of the sensory nerves and the special senses. No matter how many collections of gray matter may be interposed along the course of the nerve-fibres which convey these impressions to the cortex (each of which may possibly help to modify them), there is no argument which has yet been advanced which tends to overthrow this general law. Every image cast upon the retina, every sound-wave which enters the external ear, every odoriferous particle which reaches the nose or is placed upon the tongue, every manner of form by which we are brought into direct or indirect relation with surrounding objects during life, becomes a *conscious impression* only by affecting in some unknown way the

¹ This lecture was published in the *Journal of Nervous and Mental Diseases*, April, 1883.

cells of the cerebral cortex. Here, the image thrown upon the retina becomes to our mind the picture actually seen ; the sound-wave becomes the musical note ; the contact of the odoriferous particle is transformed by the brain-cells found in its external gray matter into a sense of smell or of taste ; objects become recognized as smooth or rough, hard or soft, heavy or light, only when these silent workers become thrown into activity by some sensory impulse carried to the convolutions of the brain by means of nerve-fibres.

We have reason to believe that the fibres of the optic nerve reach the gray matter of the convolutions of the cerebrum by different routes, and that each bundle meets (somewhere in its course) an interrupting mass of gray matter, with the cells of which the nerve-fibres become associated, and from which cells they are subsequently prolonged to those of the cortex. This is the common method of arrangement of all nerve-fibres, after they enter the substance of the brain or spinal cord, to which the optic fibres are no exception. The interrupting cells of the optic fibres are comprised chiefly within the optic thalami, the corpora geniculata, and the corpora quadrigemina. Stilling believes that a bundle of fibres can be traced to the *corpus subthalamicum*, and another to the *medulla oblongata*. The so-called "*basal optic ganglion* of Meynert" is thought by some to be also connected with a slender fasciculus of the nerve.

When speaking of these interpolated masses of gray matter and their controlling action upon all impulses sent to the brain, Michael Foster makes use of the following words, which I quote on account of their applicability to the subject under consideration :

"All day long and every day, multitudinous afferent impulses from eye, and ear, and skin, and muscle, and other tissues and organs, are streaming into our nervous system ; and did each afferent impulse issue as its correlative efferent motor impulse, our life would be a prolonged

convulsion. As it is, by the checks and counter-checks of cerebral and spinal activities, all these impulses are drilled and marshalled and kept in orderly array till a movement is called for; and thus we are able to execute at will the most complex bodily manœuvres, knowing only *why*, and unconscious or but dimly conscious *how*, we carry them out."

The study of the course of the individual fibres of the optic nerve in the region of the optic chiasm (Fig. 1) is rendered particularly difficult by the curved direction which they take; hence the relative proportion of the longitudinal and decussating bundles is still a subject of dispute among authorities upon that subject. Stilling states that inter-retinal fibres, which have no cerebral connection, can be demonstrated, while other authors deny it. Some assert that all of the fibres, which are prolonged into the optic tract, decussate in man, as they are known to do in the lower vertebrates and some mammals, but pathological observation tends to confute this view. Charcot advances the somewhat attractive theory that those fibres of the optic nerve which do not decussate at the chiasm are continued along the optic tract of the corresponding side and eventually decussate, probably within the substance of the corpora quadrigemina, after which they are continued into the internal capsule of the opposite hemisphere. He sustains this theory on pathological grounds; since many recorded cases, where lesions of the internal capsule have been associated with symptoms referable to the optic nerve, have apparently demonstrated that *hemianopsia*¹ never (?) occurs. This could not be the case without a second decussation, provided the view be considered tenable that a complete decussation does not occur at the chiasm. The diagram on page 9 will make this apparent.

¹ A term used synonymously with *hemisopia*; but a preferable one, since it signifies a blindness of one-half (lateral) of the retina.

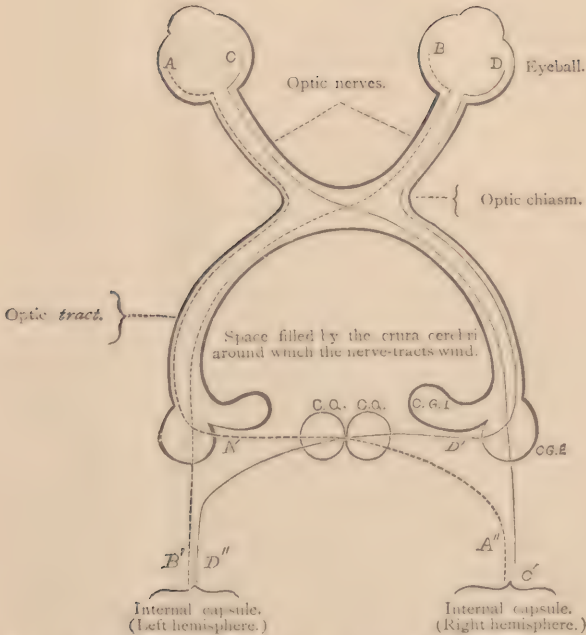


FIG. 1.—A diagram designed to show the course of fibres within the optic nerves, and some of the more important relations of the same. A, A', A'', and D, D', D'', fibres which do not cross at the chiasm, but probably do at the corpora quadrigemina. C, C', and B, B', fibres which do decussate at the chiasm. The relations of these fibres to the "internal capsule" of the cerebrum is also shown. This portion of the cerebral hemisphere is shown to be in relation with the fibres distributed only to the opposite eye: hence, lesions within it tend to produce "crossed amblyopia." The relation of bundles within the chiasm is made apparent. The fibres of the chiasm which connect the two eyes directly (inter-retinal fibres), and those which connect the two cerebral hemispheres directly (inter-cerebral fibres), are not shown, because they have no bearing upon symptoms, even if their existence is to be considered as demonstrated.

It is known that destruction of the retina in the dog gives rise to a degeneration of nerve-strands in both optic tracts. The chiasm of the cat has been divided without destroying vision, thus warranting the inference that the decussation at that point is incomplete. All the experiments which have been made to determine the relation of the cortex cerebri to vision are in favor of an incomplete decussation, because the sight of both eyes has been impaired by unilateral lesions. A large number of cases have been reported where lesions affecting one optic tract have produced hemianopsia of both retinæ.

Possibly the corpora quadrigemina preside over other functions in addition to the special sense of sight. Flourens was the first to notice that injuries of the corpora quadrigemina of one side produced peculiar phenomena, called "*forced movements*," and that the complete removal of these bodies caused inco-ordination of movement. These experiments have been repeatedly verified. In the frog, the removal of the optic lobes causes an almost entire loss of the power of *co-ordination of movements required to preserve its balance*; but it can still perform a variety of movements where co-ordination is demanded, such as swimming, leaping, etc. Schiff has attributed these effects, however, to injury of deeper parts (*crura cerebri*). We have already considered phenomena which are somewhat similar when the cerebellum was under discussion; and we have as yet no positive knowledge of the physiological connections between the optic lobes and the cerebellum.

The sense of sight has a marked effect upon co-ordination of movement, as we all know. Dizziness often follows the close inspection of a water-fall, or the rapid flight of objects before the eyes. The effect of extreme elevation from surrounding objects frequently produces marked disturbances of equilibrium. These facts seem to sustain the belief that the optic fibres must be closely associated with the cerebellum, pons Varolii, or *crura*,

and the discovery of Flourens is an additional argument in its favor.

Finally, it is believed by some that a centre which presides over the secretion of sweat is situated somewhere in the region of the optic lobes.

EFFECTS OF LESIONS OF THE OPTIC CENTRES AND OPTIC NERVES.

In connection with the discussion of the corpora quadrigemina and the probable course and distribution of the nerves of sight, it seems to me an appropriate time to mention some interesting phenomena pertaining to vision which have an important bearing upon the localization of intracranial lesions. Before doing so, however, it will be necessary to hastily review a few important facts which are essential to a complete understanding of the subject. The optic apparatus may be said to comprise the following parts :

1. *Certain cortical centres*, which act as the *interpreters* of visual sensations transmitted to the convolutions by means of the nerve-fibres within the white substance of the cerebral hemispheres. These centres probably transform all impulses (which start originally as retinal impressions) into *conscious visual perceptions*.

2. *Nodal masses of gray matter*, with which the optic nerve-fibres are intimately associated before entering the white substance of the cerebral hemisphere. These masses include the corpora quadrigemina, the corpora geniculata, the corpus sub-thalamicum, the optic centre of the thalamus (Lays), the basal optic ganglion (Meynert), and probably some centres situated within the medulla oblongata. It is not possible to speak with positiveness concerning the seat of all the interpolated

masses of nerve-cells associated with the optic nerve-fibres. Possibly some important ones may have been omitted, whose existence and function will be clearly demonstrated by future research. These interrupting ganglia probably exercise a *modifying influence* of some kind upon the impulses which are conducted to them from the retinae; and subsequently allow them to pass to the cells of the cerebral convolutions so altered or *materialized* as to be readily transformed into conscious perceptions of external objects recognized by the eyes. It is not known what the special function of each of these interrupting masses is, nor can it be determined except through a more complete knowledge of cerebral architecture and pathology than we now possess.

3. *Nerve-fibres within the optic nerves* and the *optic tracts*, the latter being the prolongation of the former behind the chiasm (see Fig. 1). These fibres convey all impressions made by objects external to the body upon the retinae by means of the organ of sight, to the interrupting masses of gray matter mentioned above. The waves of light, which enter the pupil and fall upon the retina, create in the structural elements of that membrane (probably in the so-called "rods" and "cones of Jacob") impulses which are conveyed by means of the optic fibres to the interrupting ganglion-cells, and then to the convolutions of the cerebral hemisphere where these impulses become sight-impressions. It is evident, therefore, that anything which tends to interfere with the perfect conducting power of these fibres will impair the power of accurate conception of external objects revealed to us by means of vision, because the cortical centres are cut off from their retinal connections; hence the study of the course of the nerve-fibres and the relations of the nerve-tracts to surrounding parts becomes of vital importance to the advanced neurologist (the diagnosis of many cerebral and intercranial lesions resting entirely or in part

upon optic phenomena which are to be interpreted from an anatomical standpoint alone).

4. *The retina, and its various structural elements.* This membrane constitutes the peripheral portion of the nervous optic apparatus. It is the only place in the body where the nervous system is so exposed as to admit of a direct examination, since we can see it by aid of the ophthalmoscope, and thus study its diseased conditions as well as its appearance in health. Physiologically, it is to be considered as the sensitive plate from which the details of outline and color of external objects, are telegraphed to the convolutions of the cerebrum. Many of the wonders of its construction were given you during the previous winter's course. Time will not now permit me to repeat them.¹

The experiments of Flourens, already quoted, first demonstrated that a *crude sense of vision* remains in animals which have been deprived of their cerebral convolutions above the level of the corpora quadrigemina, and many subsequent observers have attested to the accuracy of his conclusions. These experiments point to some functions within the masses of gray matter that are associated with the optic fibres, which bear a close analogy to those of the cortical cells of the so-called "visual area" of the hemispheres. We are forced to accept the view that these ganglionic masses take cognizance of visual impulses in an imperfect way, although the cerebral convolutions seem to be essential to a complete transformation of visual impulses into sight-perceptions. Section of the optic fibres after they leave the brain invariably destroys sight, thus proving that the retina itself has no inherent power of interpreting visual impressions which are cast upon it.

¹ See pages 103 to 146 of the author's work, *The Applied Anatomy of the Nervous System*. New York: D. Appleton & Co. 1881.

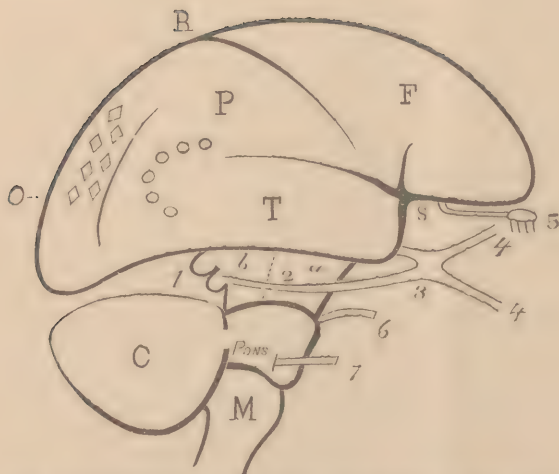


FIG. 2.—A diagram designed to show some of the relations of the optic nerve-fibres to surrounding parts. F, frontal lobes of cerebrum; P, parietal lobe; T, temporo-sphenoidal lobe; S, fissure of Sylvius; R, fissure of Rolando; O, occipital lobe; C, cerebellum; M, medulla oblongata; 1, corpora quadrigemina; 2, optic tracts; 3, optic chiasm; 4, optic nerves; 5, olfactory nerve; 6, motor oculi nerve; 7, trigeminus nerve; *a*, basis cranii; *b*, tegmentum cranii. The circles in the parietal lobe represent the *cortical visual centres of Ferrier*; the diamonds in the occipital lobe, the *cortical visual centres of Munk*. The cerebellum and pons Varoli are shown as if separated from the cerebrum, in order to make the relations of the crus to the optic tracts apparent.

Now, from what has been stated, we can classify lesions which may affect or destroy the visual function as follows :

1. *Lesions of the retina*, or of some of the other structures of the eye which prevent the formation of images within it.

2. *Lesions of the optic nerve*, anteriorly to the chiasm, at which point the decussating fibres have crossed each other.

3. *Lesions of the optic tracts and the chiasm*, or of parts so adjacent to them as to create pressure upon the optic fibres.

4. *Lesions of those ganglionic masses* whose connection with the optic fibres has been demonstrated by anatomical or pathological research.

5. *Lesions of certain regions of the cortex cerebri*, which have been shown to be in intimate association with vision.

6. *Lesions of the internal capsule* of the cerebrum ; or of such parts of the medullary centre of each hemisphere as contain fibres connected with the "visual area" of the cortex.

The *first set* of causes of impairment of vision belongs properly to the province of the oculist rather than of the neurologist, although there is one condition which should always be sought for when cerebral disease is suspected, viz., *neuro-retinitis*, or the so-called "*choked disc*." The evidences of this condition are afforded by the ophthalmoscope alone, because vision is not impaired in the early stages. Its existence is recognized early by tor

tuosity of the veins of the fundus of the eye, swelling of the optic nerve, and obscureness of the margin of the disc ; later, the outline of the disc becomes unnaturally sharp and distinct, the nerve atrophies, the vessels become very small, the fundus is unnaturally pale, and vision is impaired. This condition is always (?) bilateral, although it is not uncommon to note a marked difference in the severity of the changes in the two eyes. Special attention is called to this disease of the eye, because it is now considered as one of the most reliable signs of conditions of the cerebrum which tend to produce a *gradually increasing pressure*, particularly of tumors ; and in the second place, because its existence is liable to be overlooked, since vision is not impaired early. The various phenomena which are due to paresis of ocular muscles,¹ and which have often the most positive value in definitely localizing cerebral disease, cannot be considered under this set of symptoms or in this connection, because they do not govern in any way the sense of sight, although they assist the eye to focus images of objects upon the retina. My friend, Dr. W. C. Ayers, has lately made a valuable contribution to medical literature in the form of a brochure upon the value of the ophthalmoscope as an aid in general diagnosis,² which may well be consulted by all who desire the latest information in regard to the utility of this instrument, and the intimate relationship which exists between the eye and the body, as revealed by clinical study.

The *second set* of causes of impairment of vision (lesions of the optic nerve anteriorly to the chiasm) includes chiefly those *conditions within the orbit* which create pressure upon, or destruction of, the optic nerve after it leaves the cavity of the cranium. It is evident from the diagram (Fig. 1) that the impairment of sight in

¹ These will be found by consulting the author's work, *The Applied Anatomy of the Nervous System*. New York, 1881.

² *American Journal of the Medical Sciences*, October, 1882.

this case will be confined exclusively to one eye. The phenomena produced by disease within the cavity of the orbit upon sight must, of course, depend upon the amount of injury done to the optic nerve. *Blindness of one eye* indicates, as a rule, some exciting cause outside of the cavity of the cranium; if it occurs in connection with hemiplegia, or hemianæsthesia, however, a lesion of the internal capsule may be suspected, as shown in Fig. 1. Remember that either amblyopia or total blindness of one eye never occurs in connection with pressure upon the optic nerve-fibres either at the chiasm or behind it, and that some form of paralysis must coexist with these symptoms if the internal capsule be diseased.

We come now to the *third set* of causes, viz., lesions of the optic tracts and chiasm. This set includes not only actual lesions of the nerve, but also pressure-effects exerted upon the optic fibres by lesions of adjacent structures. Before we pass to the consideration of the diagnostic symptoms of this condition, it is important that we review some of the relations of the optic chiasm and the optic tracts.

If we trace the optic nerve fibres from behind forward, we find that the *optic tracts* appear to arise from the optic thalami, the superior quadrigeminal bodies, and the corpora geniculata. As they leave the under surface of the thalami, they make a sudden bend forward and curve around the crura cerebri in the form of a flattened band (Fig. 1). At their anterior portions the tracts become closely attached to the crura, and, in the region of the tuber cinerium, an accession of fibres to the tracts may be demonstrated. Before the chiasm is reached the tracts become more cylindrical in form.

The *optic commissure*, or *chiasm*, is about one-half of an inch long in its transverse measurement, and lies upon the olivary process of the sphenoid bone. The internal carotid arteries lie in close relation with it at the sides, and the anterior cerebral arteries, with their com-

municating branch, are so disposed as to constitute what might be called a *loop* about the optic nerves. The clinical bearing of this fact will be discussed later. Henle reports a few remarkable instances where the chiasm was wanting, the optic tracts being continued without interruption to the eyeball of the corresponding side; but these abnormalities are so rare as to be of no practical importance from a clinical standpoint.

We are now able to study the diagram which I draw upon the blackboard, and to properly interpret the clinical deductions which may be drawn from it. It is intended to portray the effects of localized pressure upon the optic chiasm and optic tracts, as well as those of destructive lesions of the same and of the "internal capsule" of the brain. Figs. 1 and 2 will assist us in following the details of this diagram, as they are somewhat similar.

Now, this diagram is admirably adapted to portray the mechanism of one peculiar symptom, and the use to which it may be employed by the neurologist in definitely determining the seat of the disease-lesion which is creating it. I refer to "*hemianopsia*," or *blindness of one lateral half of the retina*. The term "*hemioptia*" is often employed to express the same condition, although it is to my mind a poor one, since it simply means "half-vision," and thus fails to express the idea intended.

[The description of the various types of hemianopsia which followed in the course of the lecture has been omitted, because the text which accompanies the diagram covers all essential points. The author has also discussed the clinical bearings of this subject very fully in his work, "*The Applied Anatomy of the Nervous System*," D. Appleton & Co., New York.]

The following steps are commonly employed to detect the existence of this symptom: Request the patient to close one eye by pressing the lid down with the finger, and to so direct the open eye as to concentrate its gaze

upon some fixed object near to it. [I usually hold up the forefinger of my own hand within a foot of the patient's open eye, and tell him to look steadily at it.] Having done this, take some object which is easily seen (such as a sheet of white paper) in the unemployed hand, and move it to the right and left of the object upon which the patient is gazing, and also above and below the object, asking the patient, in each case, if the two objects are seen simultaneously and with distinctness, and notice upon which side of the fixed object the patient cannot perceive the moving object. It is self-evident that the retina is blind upon the side opposite to that upon which the moving object is lost to sight (see Fig. 3).

The most common form of hemianopsia is that in which the *nasal half* of one eye and the *temporal half* of the other is blind; this condition being the result of pressure upon, or actual destruction of, one of the *optic tracts*.

When the *chiasm* is affected, we meet the *bi-nasal* type.

There is still one more form which is occasionally encountered, viz., the *bi-temporal* type. This has been interpreted by an autopsy made upon a case entrusted to the care of Professor H. Knapp, of this city. It must be evident that the chances would be extremely small of ever encountering a bi-lateral lesion which would affect only those fibres of the optic chiasm, or optic tract, which supply the temporal half of each retina, and, at the same time leave the decussating fibres intact. How, then, are we to account for the fact that this form is sometimes met with? In the preceding portion of this lecture I called your attention to a peculiar arrangement of the arteries in the region of the optic chiasm. Now, it has been shown that atheromatous degeneration of the "circle of Willis" (a peculiar arrangement of blood-vessels at the base of the brain) so impairs the elasticity of the arteries as to create a type of injury to the chiasm, so limited in its extent as to impair only the fibres distrib-

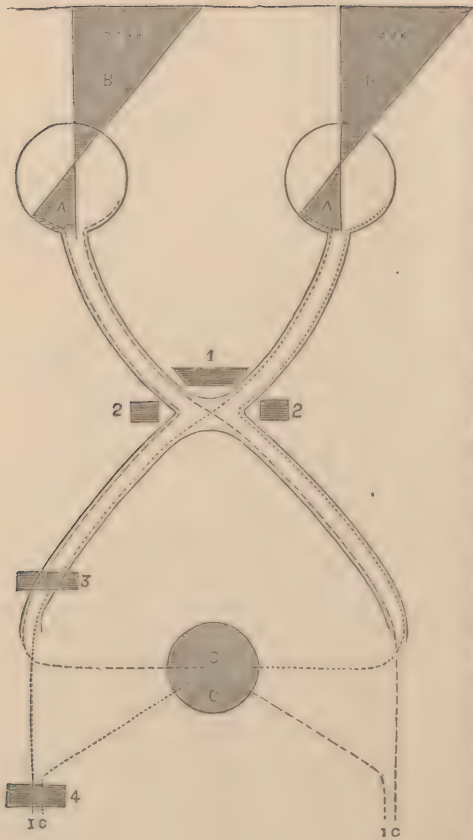


FIG. 3.—Diagram explicative of hemiopia (Seguin). The shaded intra- and extra-ocular parts, A and B, indicate the obscuration in right lateral (or homonymous) hemiopia, as caused by lesion 3, so placed as to destroy one optic tract. In that tract are two-sets of nerve-fibres, one represented by a dotted line supplying the nasal half of right retina, the other fibres by a broken line supplying the outer or temporal half of the left eye. As visual lines cross in the eye the obscuration of the half fields is the opposite. Lesion No. 1, anterior to chiasm, produces blindness of inner half of each retina, and consequently in temporal hemiopia. Lesion No. 2, pressing upon the sides of the chiasm, injures fibres supplying the temporal half of each retina, and cause in nasal hemiopia. C C, corpus quadrigeminum, in which Professor Charcot believes a second partial decussation takes place. I C, internal capsule containing, on Charcot's hypothesis, all the fibres coming from the eye of the opposite side. 4. Lesion of internal capsule injuring all the fibres connected with the right retina, and causing amblyopia of the right eye.

uted to the temporal halves of the retinae, and thus to create bi-temporal hemianopsia.

We may, therefore, summarize the clinical significance of this peculiar form of blindness as follows :

(a.) The *homonymous* or *crossed variety* indicates lesions affecting the optic tract.

(b.) The *bi-nasal variety* indicates a lesion pressing upon the central portion of the chiasm.

(c.) The *bi-temporal variety* indicates atheromatous degeneration of the circle of Willis. Possibly (?) symmetrical lesions of the outer part of the chiasm might also cause it. I am not aware that the view of Charcot, that a decussation of the optic fibres takes place within the substance of the corpora quadrigemina, is as yet sustained by a recorded case of bi-temporal hemianopsia produced by a circumscribed lesion within the optic lobes.

(d.) Finally, lesions of the *internal capsule* are often associated with *amblyopia*, or indistinct vision confined to one eye.

The bi-nasal, and also the bi-temporal varieties, are due (as a rule, at least) to lesions confined to the *anterior fossa* of the cranium; hence we sometimes find the *olfactory nerve* of the side corresponding to the seat of the lesion simultaneously affected, and creating anosmia (loss of smell) with or without subjective odors.

If the lesion be situated within the *middle fossa* of the cranium, the *optic tracts* will be affected, thus causing crossed hemianopsia; while the *motor nerves of the eye* may be simultaneously pressed upon, as they pass through that fossa on the way to their foramen of exit from the cranium (the sphenoidal fissure), thus produ-

cing more or less impairment of the movements of the eyeball of the same side. The value of these complications cannot be over-estimated, when they exist, because they are of the greatest aid in diagnosis, and often enable the skilled anatomist to positively determine the seat of the lesion.

The *fourth set* of causes of impairment of vision (according to the classification which we have previously adopted) comprises all diseased conditions which are limited exclusively to those ganglionic masses through which the optic fibres pass in order to reach their connections with the convolutions of the cerebrum. If we confine ourselves to this strict limitation, we are forced to admit that little can positively be said respecting them which will bear upon intracranial diagnosis, because, to my knowledge, there are no recorded cases where evidences of cerebral disease have been confined exclusively to these regions. There are some symptoms, however, that may coexist with disturbances of vision when lesions exist in the region of the middle fossa of the skull; these may prove of assistance in deciding as to the seat of the lesion. Among them may be enumerated: 1. *Crossed paralysis* of the "*third nerve and body*" type, a condition characterized by hemiplegia and paralysis of the motor-oculi nerve of the opposite side. 2. *Crossed paralysis* of the "*olfactory nerve and body*" type, a condition characterized by hemiplegia and loss of smell in the opposite nostril. 3. *Hemiplegia*, or loss of the power of voluntary motion in one lateral half of the body. 4. *Hemianesthesia*, or a loss of sensation in one lateral half of the body. 5. *Ataxic symptoms*, indicating an impairment of co-ordination of muscular movements.

The first of these points positively to a *lesion of the crus cerebri*, if unattended by other symptoms. But, if evidences of disturbance of the optic tract (crossed hemianopsia) exists simultaneously with this form of crossed paralysis, it indicates that the lesion is large

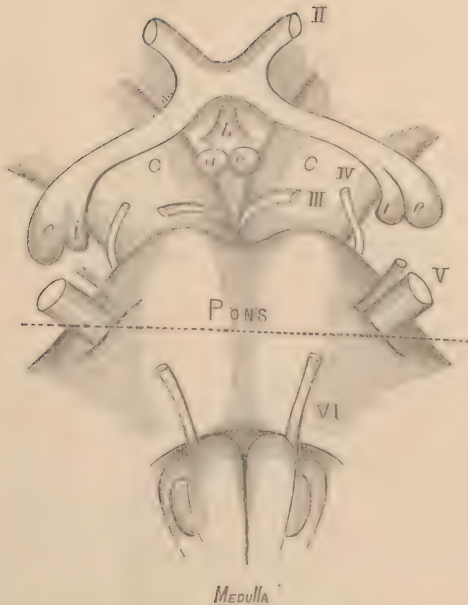


FIG. 4.—A diagram of the base of the brain, designed to show the parts adjacent to the optic nerve tracts and chiasm. The nerves are represented by their respective numbers: II., optic; III., motor oculi; IV., trochlears; V., trigemini; VI., abducent; C, crus cerebri of each hemisphere; b, internal geniculate, the pituitary body being cut off to show the optic chiasm; a, the corpus callosum (mamillary tubercle); e, external geniculate body; d, internal geniculate body. The dotted line which crosses the pons Varoli, connecting the roots of the fifth nerves, is Gasser's line, an important guide, since lesions of the pons in front of it causes "crossed field paralysis." Lesions in the region of the crura may involve the third and second nerves simultaneously. Lesions about the chiasm may press upon the corpus striatum within the mass of the cerebrum. The crus comprises both the motor and sensory tracts of the cerebrum (see Fig. 5).

enough to interfere also with the optic nerve as well as the motor-oculi fibres within the crus and the motor tract of the crus. The symptoms of this variety of crossed paralysis¹ are so well defined as to render it almost impossible to mistake them.

The second condition may occur when the lesion is sufficiently large to create pressure upon the nucleus caudatus or other motor parts of the brain, thus causing hemiplegia of the opposite side; and, at the same time, to injure the olfactory nerve, thus causing anosmia (loss of smell) in the nostril of the corresponding side. Of course the optic tract or chiasm must be involved simultaneously when hemianopsia also exists. The tests for anosmia² have already been given in previous lectures.

Hemiplegia may occur in connection with hemianopsia when the lesion is of sufficient size to affect any part of the so-called "*motor tract*" simultaneously with the optic nerve-fibres. The motor paralysis is on the side opposite to the lesion, because the fibres of the motor tract decussate at the lower part of the medulla. Flechsig has shown that, in rare cases, *exceptions* to this general rule are to be explained by an abnormality in the decussation of the motor fibres. Hemiplegia is seldom observed in connection with hemianopsia alone, since the olfactory, motor-oculi, trigeminus, and facial nerve-roots are especially liable to be simultaneously involved (see Fig. 2). This explains the mechanism of the four varieties of "crossed paralysis" which are encountered, the hemiplegia being on the side opposite to the lesion, and the symptoms produced by paralysis of the cranial nerve being confined to the side corresponding to the lesion.

Hemianesthesia indicates some disturbance of the nerve-fibres of the so-called "*sensory tract*," the loss of

¹ See author's work on The Applied Anatomy of the Nervous System.

² Op. cit.

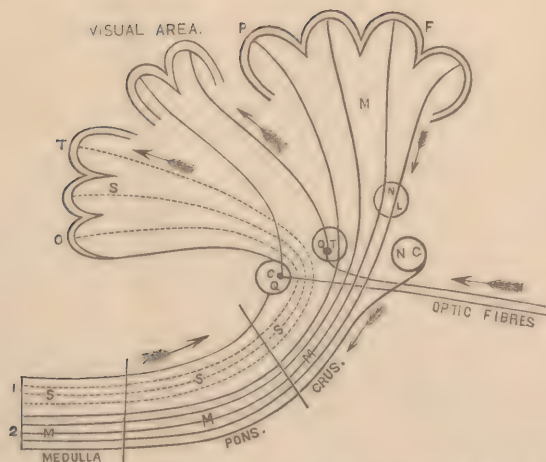


FIG. 5.—A diagram designed to show the general course of fibres in the "sensory" and "motor tracts," and their relation to certain fasciculi of the optic nerve-tracts (modified from Seguin). S, sensory tract in posterior region of mesocephalon, extending to O and T, occipital and temporal lobes of hemispheres; M, motor tract in basis cruris, extending to P and F, parietal and (part of) frontal lobes of hemispheres; C Q, corpus quadrigemium; O T, optic thalamus; N L, nucleus lenticularis; N C, nucleus caudatus; 1, the fibres forming the "tegmentum cruris" (Meynert); 2, the fibres forming the "basis cruris" (Meynert); α , fibres of the optic nerve which become associated with the "optic centre" in the optic thalamus, and are subsequently prolonged to the "visual area" of the convolutions of the cerebrum; β , optic fibres which join the cells of the "corpora quadrigemina," and are then prolonged to the visual area of the cerebral cortex.

sensation being confined to the lateral half of the body opposite to the lesion which causes it, because the sensory fibres decussate in the spinal cord. In cerebral hemianæsthesia there is more or less insensibility to touch, pain, and temperature, and also abolition of muscular sensibility with complete retention of electro-motor contractility. The mucous membranes of the eye, nose, and mouth are also anæsthetic. Now the upper portion of the sensory tract lies in the posterior regions of the crus cerebri and the internal capsule and crus, and is in close relation with the posterior basal ganglia. The fibres of the optic tract may be likewise affected simultaneously with lesions of the following parts: the crus, the internal capsule, the optic thalamus, the corpora quadrigemina, the geniculate bodies, and the medulla. It has been already stated that lesions of the internal capsule are often associated with *amblyopia*, but not with hemianopsia. Fig. 1 will make the probable explanation of this fact intelligible. Our ability to definitely locate lesions of the sensory tract, or of the ganglia connected with it, is, as yet, imperfect. It is only by the careful study of associated symptoms that conclusions can be arrived at.

Ataxic manifestations, occurring in connection with evidences of impairment of the sense of sight, open a wide field for speculation. The proximity and intimate structural relations of the cerebellum with the optic lobes, basal ganglia, crus, and medulla, suggest the possibility of cerebellar lesions when these two symptoms are present to a marked degree. The subject is too complex for discussion here. It will be more intelligible after the cerebellum has been considered.

The *fifth set* of causes of impairment of vision previously tabulated, will now be considered. Within the past few years the attention of physiologists has been directed, by some remarkable results of experimentation upon the convolutions of the cerebral cortex, toward the view that

certain convolutions of the cerebrum were essential to perfect visual perceptions. To Flourens and some of the older observers, who had remarked that the removal of portions of the hemispheres, or serious injury to them, created blindness, the loss of sight appeared to be temporary. The fact (?) was explained in various ways, until Goltz called attention to the error of supposing that no permanent imperfections of vision remained after extensive injuries to the cerebral hemispheres. This author showed that the permanent results of such injuries might escape notice unless special care was used in the examinations of the animal. The peculiarities of the permanent impairment of vision are manifested only when the animal is subjected to tests which had been invariably potent before the cerebral injury. Thus the dog, from which portions of the cerebral hemispheres had been removed, fails to recognize his food by sight; when he is threatened by a whip he is not cowed; he is no longer affected by objects which caused him to be violently excited before the mutilation; he makes no response to the extension of the hand of his master for the paw; and yet this animal can see to avoid objects and to perform all varieties of movement, as well as in his natural condition. Another striking characteristic of this impairment of sight is, that under educational exercise recovery takes place. The dog may again be taught to fear castigation and to shrink at the sight of the whip; to recognize his food; to obey the motions of his master's hand, etc.

Two interpretations of these phenomena have been suggested. The first is, that the animal has imperfect visual perceptions, so that objects appear misty or as if seen through a gauze. Goltz suggests that they may appear as if all the colors were washed out, thus depriving food, dress, etc., of their characteristic appearances. The second interpretation supposes that the memory of past visual impressions is effaced, so that the animal for-

gets the pain of the lash, the taste of the food, the features of his master, the tricks which have been laboriously taught him. The first view is that of Goltz, who considers that the animal has to learn to use his imperfect visual perceptions before his intellectual faculties (which are presumed to be unimpaired) can respond to them in a proper way. The second view is that of Munk, who speaks of this form of imperfect vision as "psychical blindness," in contrast to "absolute blindness," which is the result of destruction of the optic fibres. The condition of the animal resembles that of the new-born. Retinal impressions have no associations connected with them. During the period of recovery the animal has to acquire a new memory, as it were.

With this distinction clearly in mind, we are prepared to discuss the views of Ferrier, Goltz, Munk, Luciani, Tamburini, Yeo, Dalton, and others respecting the exact seat of the visual centres within the cortex cerebri.

Goltz, in his experiments upon dogs, was unable to recognize any distinct areas which presided exclusively over visual impressions. He insists that disturbances of general sensibility accompanied the impairment of vision produced by destruction of the convolutions, and that the results depended upon the amount of brain-substance removed or destroyed. He found, however, that the locality operated upon influenced the phenomena which followed, and that recovery would take place if the injury was not too extensive. Goltz destroyed the brain by making a hole, or a number of them, through the skull, and using a *forcible stream of water* to wash away the brain-substance. The faults of the method may account for the negative results obtained by it.

Ferrier investigated the subject chiefly upon the monkey tribe (the nearest approach to the human race) and arrived at conclusions of a more positive character. This observer was led to adopt a more certain way of limiting the injury done to the cortex than that of Goltz. His

conclusions may be thus summarized : When the "angular gyrus," or a convolution of the parietal lobe, so called from its shape, since it forms a sharp angle (see Fig. 2), was destroyed upon one side only, the vision of the *opposite eye* was destroyed for a time, but it eventually regained its powers. If the angular gyrus of each hemisphere was simultaneously destroyed, the *loss of sight was permanent* and both eyes were equally affected. Hence it would appear that each hemisphere is in some way connected with both eyes, because unilateral destruction of this convolution does not create permanent blindness, as it would do if the opposite hemisphere did not come to its relief. Dalton has lately confirmed the views of Ferrier by experiments made upon dogs, thus tending to confute the view of Goltz that the effects of cortical lesions depend more on their extent than on their position. The animals operated upon by this observer remained permanently blind, although the lesion was unilateral.

Munk has confined his experiments chiefly to the occipital lobes of the cerebrum, and has endeavored to demonstrate the existence of a "visual area," differing in position and of much wider extent than that of Ferrier (Fig. 2). He maintains that certain parts of this region can be shown to preside over *limited portions* of the retina, and that blindness of circumscribed spots in the retina can be artificially produced. He states that the "absolute blindness" thus created is commonly associated with "psychical blindness," from which the animal may recover by proper exercise and training, provided the whole visual area is not destroyed. This author attributes the recovery to a deposition of new visual experiences in the rest of the visual area.

Some observations which have lately been made in support of the view that the occipital lobes of the cerebrum are more directly associated with the mental faculties than the frontal lobes are of special interest to those who agree with Munk regarding the seat of the visual

area of the cortex. At present, however, the subject must be regarded as unsettled.

The *fifth set* of causes of impairment of vision, previously tabulated, has been discussed in part in connection with the others. We have a mass of clinical as well as experimental evidence to show that destructive lesions situated within the *posterior one-third* of the internal capsule cause *hemianæsthesia* on the opposite side of the body. As regards vision, the symptoms which sometimes exist are especially noteworthy. There appears to be developed on the anæsthetic side a partial blindness of the eye (*amblyopia*), and the *field of vision for color* is remarkably contracted, as first pointed out by Landolt. In the normal eye the field for blue is the largest; next comes that for yellow; then orange, red, green, and violet have fields of gradually diminishing size, the last being perceived only by the most central parts of the retina. Now, in connection with hemianæsthesia caused by cerebral lesions, the perception of violet first disappears, then for green, and later for orange. In some cases yellow and blue can be perfectly recognized; but in the higher degrees of anæsthesia all colors merge into an uniform sepia tint. Another important fact has been pointed out by Landolt, viz., that the eye on the same side as the lesion participates, though to a less extent, in the loss of color-perception.

Now, if the eyes be examined with the ophthalmoscope, no evidences of organic lesion of the retina or degeneration of the optic nerve can be detected until the disuse of the organ has induced atrophic changes. It is noteworthy that hemianopsia does not occur, a fact which Fig. 3 will help to explain. From statements which have been made in preceding pages, we are forced to conclude that this peculiar type of blindness is never caused except by lesions which are situated or act below the cortex. It is more commonly met with in connection with hemiplegia than hemianopsia. The effects of

lesions of the internal capsule upon other nerves of special sense will be considered later.

Clinical deductions drawn from preceding pages.—*Amblyopia* of one eye can result from lesions involving the optic nerve *in front of the chiasm*, or from *lesions of the internal capsule*. If from the latter, the field for color-perceptions will be found to be markedly contracted or color-vision will be wanting; both eyes may be affected, the most marked changes being found, however, in the eye opposite to the seat of the lesion. We have not sufficient data for positive clinical deductions respecting lesions of the visual area of the cortex in man. The blindness of the opposite eye appears to be absolute, while it lasts, in all animals upon whom the experiment has been tried.

Hemianopsia only occurs when the *optic tracts* or the *optic chiasm* are pressed upon or destroyed by lesions in the region of the base of the cerebrum. It is evident, therefore, that the trephine cannot afford relief of this symptom. When syphilitic gummata may be suspected, the prognosis is extremely favorable if active treatment be employed. The variety of hemianopsia indicates the seat of the lesion with great exactness.

If *paralysis* (in any of its forms) coexist with hemianopsia, a valuable guide will often be afforded in determining the extent of the lesion.

Crossed paralysis of the "olfactory nerve and body type" indicates a localized pressure which is chiefly exerted upon parts *within the anterior fossa of the skull*. The motor tract is probably involved by upward pressure upon the caudate or lenticular nucleus, or the fibres of the internal capsule; thus accounting for the hemiplegia of the opposite half of the body. The olfactory nerve, which lies near to the optic chiasm, is affected by pressure in the downward direction, and the optic chiasm or tract may be simultaneously involved; hence, a loss of smell in the nostril on the same side as the lesion may

coexist with some form of hemianopsia, as well as with a crossed hemiplegia.

Crossed paralysis of the "motor-oculi nerve and body" type indicates a *lesion situated within the crus cerebri*. If hemianopsia be present in connection with this condition, it proves conclusively that the optic tract, which lies in close relation with the crus, is simultaneously affected by the lesion. We find, therefore, that the eye on the same side as the lesion is blind in its temporal half if the optic tract be involved; that it can no longer be turned toward the nose or made to act in parallelism with the opposite eye; that the pupil is dilated; and that the upper eyelid droops over the eyeball, giving it a sleepy appearance. On the side opposite to the lesion the eye is blind in its nasal half, and the body is hemiplegic. There are few conditions which are of greater clinical importance than this type of crossed paralysis, because the seat of the lesion is positively indicated. ‡

Choked disc is a common symptom of lesions of the base of the cerebrum, and of any intracranial disease which produces a gradually increasing pressure. It is specially diagnostic of tumors. It is not associated with impairment of vision until late, so that it is often unsuspected when present. The ophthalmoscope is necessary for its detection. It may coexist with hemianopsia, and is always bilateral. It is a positive contra-indication to trephining.

Lesions at the base of the skull may *cross the mesial line*, and still involve only one optic tract. If this occurs, the hemianopsia will be accompanied by other symptoms of diagnostic importance, no longer confined to one side. Double anosmia, general paresis or complete paralysis, general anæsthesia, and paralytic symptoms referable to both eyeballs might be thus produced. Lesions of this character are more liable to affect the chiasm of the optic nerves than the optic tracts; in either case, however, hemianopsia would result, and its

type would be a reliable guide to the seat of pressure (see Fig. 4).

Crossed paralysis of the "facial nerve and body type" is not as liable to coexist with hemianopsia as the two forms previously mentioned. The reason for this is a purely anatomical one. The symptoms of facial paralysis are too involved to be given here in detail.¹

Uncomplicated hemianopsia indicates that the effect of the lesion are confined to the optic tracts of chiasm, and that no pressure-effects are exerted upon the motor or sensory projection tracts, or adjacent nerves.

Aphasia sometimes coexists with hemianopsia. I have met with two instances of this kind. In one there was slight paresis of the left side, tending to prove that aphasia can occur with lesions involving the right hemisphere. Both were cured with specific treatment. We must attribute the development of this complication to pressure upon parts in the neighborhood of Broca's centre.

Lesions confined to the crus cerebri seldom create impairment of any of the special senses excepting that of the sight. These cases are not associated with impairment of intellect or of speech. It has been claimed that severe lesions cause paralysis of the bladder, but I have never encountered it. Many points of interest pertaining to lesions of the crura will be considered later in the course.

¹ They are fully discussed in the author's work on *The Applied Anatomy of the Nervous System*.

